WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Wednesday, June 21, 2006

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,DWPI; PLUR=NO; OP=ADJ		
☐	L8	US 20040142346 A1	2
☐	L7	((dsrna or sirna) near5 (pna or aminoethylglyc$)) and ((@pd<20030829) or (@ad<20030829))	26
☐	L6	((dsrna or sirna) with (pna or aminoethylglyc$)) and ((@pd<20030829) or (@ad<20030829))	53
	DB=USPT; PLUR=NO; OP=ADJ		
☐	L5	((dsrna or sirna) with (pna or aminoethylglyc$)) and ((@pd<20030829) or (@ad<20030829))	8
☐	L4	l3 and ((dsrna or sirna) with pna)	0
☐	L3	pna and (@pd<20030829) and (dsrna or sirna)	18
☐	L2	isis.as. and pna and (@pd<20030829) and (dsrna or sirna)	2
☐	L1	6228642.pn.	1

END OF SEARCH HISTORY

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1635jxs

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS 4	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS 5	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 6	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 7	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 8	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 9	MAR 22	EMBASE is now updated on a daily basis
NEWS 10	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS 11	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS 12	APR 04	STN AnaVist $500 visualization usage credit offered
NEWS 13	APR 12	LINSPEC, learning database for INSPEC, reloaded and enhanced
NEWS 14	APR 12	Improved structure highlighting in FQHIT and QHIT display in MARPAT
NEWS 15	APR 12	Derwent World Patents Index to be reloaded and enhanced during second quarter; strategies may be affected
NEWS 16	MAY 10	CA/CAplus enhanced with 1900-1906 U.S. patent records
NEWS 17	MAY 11	KOREAPAT updates resume
NEWS 18	MAY 19	Derwent World Patents Index to be reloaded and enhanced
NEWS 19	MAY 30	IPC 8 Rolled-up Core codes added to CA/CAplus and USPATFULL/USPAT2
NEWS 20	MAY 30	The F-Term thesaurus is now available in CA/CAplus
NEWS 21	JUN 02	The first reclassification of IPC codes now complete in INPADOC

NEWS EXPRESS JUNE 16 CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 23 MAY 2006.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8
NEWS X25	X.25 communication option no longer available after June 2006

Enter NEWS followed by the item number or name to see news on that specific topic.

* * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

```
FILE 'HOME' ENTERED AT 14:28:06 ON 21 JUN 2006

=> FIL MEDLINE BIOSIS CA EMBASE SCISEARCH
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 14:28:18 ON 21 JUN 2006

FILE 'BIOSIS' ENTERED AT 14:28:18 ON 21 JUN 2006
Copyright (c) 2006 The Thomson Corporation

FILE 'CA' ENTERED AT 14:28:18 ON 21 JUN 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'EMBASE' ENTERED AT 14:28:18 ON 21 JUN 2006
Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE 'SCISEARCH' ENTERED AT 14:28:18 ON 21 JUN 2006
Copyright (c) 2006 The Thomson Corporation

=> s tnf-alpha or cachectin or tnf-a or TNFA or TNFSF2 or DIF
L1      237789 TNF-ALPHA OR CACHECTIN OR TNF-A OR TNFA OR TNFSF2 OR DIF

=> s (dsrna or sirna or shrna or rnai)
L2       55887 (DSRNA OR SIRNA OR SHRNA OR RNAI)

=> s l1 and l2
L3         720 L1 AND L2

=> s l3 and (py<=2003)
   1 FILES SEARCHED...
L4         217 L3 AND (PY<=2003)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5          81 DUP REM L4 (136 DUPLICATES REMOVED)

=> s l5 and (l1 same l2)
MISSING OPERATOR L1 SAME
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s (l1 same l2) and l5
MISSING OPERATOR L1 SAME
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s (l1 (s) l2) and l5
L6          27 (L1 (S) L2) AND L5

=> s l6 and (sirna (s) l1)
L7           5 L6 AND (SIRNA (S) L1)

=> d l7 ibib abs 1-5

L7   ANSWER 1 OF 5     MEDLINE on STN
ACCESSION NUMBER:   2003565087     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 14652004
TITLE:              Cationic liposome-mediated delivery of siRNAs in
                    adult mice.
AUTHOR:             Sioud Mouldy; Sorensen Dag R
```

CORPORATE SOURCE: Department of Immunology, Molecular Medicine Group, The Norwegian Radium Hospital, 0310, Montebello, Norway.. mosioud@ulrick.uio.no
SOURCE: Biochemical and biophysical research communications, **(2003 Dec 26)** Vol. 312, No. 4, pp. 1220-5. Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200403
ENTRY DATE: Entered STN: 16 Dec 2003
Last Updated on STN: 19 Mar 2004
Entered Medline: 18 Mar 2004

AB RNA interference mediated by small interfering RNAs (**siRNAs**) is a powerful tool for dissecting gene function and drug target validation. **siRNAs** can be synthesized in large quantities and thus can be used to analyze a large number of sequences emerging from genome projects in a cost-effective manner. However, the major obstacle to the use of **siRNAs** as therapeutics is the difficulty involved in effective in vivo delivery. We used a fluorescein-labeled **siRNA** to investigate cationic liposome-mediated intravenous and intraperitoneal delivery in adult mice. We show that this simple approach can deliver **siRNAs** into various cell types. In addition, we show that in contrast to mouse cells, **siRNAs** can activate the non-specific pathway in human freshly isolated monocytes, resulting in **TNF-alpha** and IL-6 production. Taken together, the data provide a basis for lipid-mediated systemic delivery of **siRNAs** and indicate that certain **siRNA** sequences can activate the innate immunity response genes that can be beneficial for the treatment of cancer.

L7 ANSWER 2 OF 5 MEDLINE on STN
ACCESSION NUMBER: 2003480723 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14559223
TITLE: Introduction of short interfering RNA to silence endogenous E-selectin in vascular endothelium leads to successful inhibition of leukocyte adhesion.
AUTHOR: Nishiwaki Yasunobu; Yokota Takanori; Hiraoka Megumi; Miyagishi Makoto; Taira Kazunari; Isobe Mitsuaki; Mizusawa Hidehiro; Yoshida Masayuki
CORPORATE SOURCE: Department of Medical Biochemistry, Tokyo Medical and Dental University, Tokyo, Japan.
SOURCE: Biochemical and biophysical research communications, **(2003 Oct 31)** Vol. 310, No. 4, pp. 1062-6. Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 16 Oct 2003
Last Updated on STN: 18 Dec 2003
Entered Medline: 17 Dec 2003

AB Short interfering RNAs (**siRNAs**) are powerful sequence-specific reagents that suppress gene expression in mammalian cells. We report for the first time that gene silencing of endothelial E-selectin by **siRNAs** leads to successful inhibition of leukocyte-endothelial interaction under flow. **siRNAs** designed to target human E-selectin were tranfected into human umbilical vein endothelial cells (HUVEC). Western blotting analysis revealed that transfection of these **siRNAs**, but not the scrambled control **siRNA** (100nM each), attenuated E-selectin expression in HUVEC activated with **TNF-alpha** (10ng/ml, 4h) without affecting expression of

ICAM-1. Moreover, a leukocyte adhesion assay under flow (shear stress=1.0dyne/cm(2)) demonstrated that HUVEC transfected with a **siRNA** against E-selectin (siE-01) supported significantly less HL60 adhesion as compared to those transfected with the control **siRNA** (scE-01) after activation ($p<0.03$). This technique provides a powerful strategy to dissect a specific function of a given molecule in leukocyte-endothelial interaction.

L7 ANSWER 3 OF 5 MEDLINE on STN
ACCESSION NUMBER: 2003140017 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12654261
TITLE: Gene silencing by systemic delivery of synthetic **siRNAs** in adult mice.
AUTHOR: Sorensen Dag R; Leirdal Marianne; Sioud Mouldy
CORPORATE SOURCE: Department of Comparative Medicine, The National Hospital, Oslo 0310, Norway.
SOURCE: Journal of molecular biology, **(2003 Apr 4)** Vol. 327, No. 4, pp. 761-6.
Journal code: 2985088R. ISSN: 0022-2836.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 26 Mar 2003
Last Updated on STN: 2 May 2003
Entered Medline: 1 May 2003

AB In mammalian cells, RNA duplexes of 21-23 nucleotides, known as small interfering RNAs (**siRNAs**) specifically inhibit gene expression in vitro. Here, we show that systemic delivery of **siRNAs** can inhibited exogenous and endogenous gene expression in adult mice. Cationic liposome-based intravenous injection in mice of plasmid encoding the green fluorescent protein (GFP) with its cognate **siRNA**, inhibited GFP gene expression in various organs. Furthermore, intraperitoneal injection of anti-**TNF-alpha** **siRNA** inhibited lipopolysaccharide-induced **TNF-alpha** gene expression, whereas secretion of IL1-alpha was not inhibited. Importantly, the development of sepsis in mice following a lethal dose of lipopolysaccharide injection, was significantly inhibited by pre-treatment of the animals with anti-**TNF-alpha** **siRNAs**. Collectively, these results demonstrate that synthetic **siRNAs** can function in vivo as pharmaceutical drugs.

L7 ANSWER 4 OF 5 BIOSIS on STN
ACCESSION NUMBER: 2003:356987 BIOSIS
DOCUMENT NUMBER: PREV200300356987
TITLE: The PAAD/PYRIN-Family Protein ASC Is a Regulator of a Conserved Step in NF-kB Activation Pathways.
AUTHOR(S): Stehlik, Christian [Reprint Author]; Fiorentino, Loredana [Reprint Author]; Dorfleutner, Andrea [Reprint Author]; Ariza, Eugenia M. [Reprint Author]; Sagara, Junji [Reprint Author]; Reed, John C. [Reprint Author]
CORPORATE SOURCE: The Burnham Institute, La Jolla, CA, USA
SOURCE: Blood, **(November 16 2002)** Vol. 100, No. 11, pp. Abstract No. 2853. print.
Meeting Info.: 44th Annual Meeting of the American Society of Hematology. Philadelphia, PA, USA. December 06-10, 2002. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English

ENTRY DATE: Entered STN: 6 Aug 2003
Last Updated on STN: 18 Sep 2003

AB ASC ("Apoptosis-associated speck-like protein containing a Caspase recruitment domain") is a bipartite protein containing both a CARD and a PAAD/PYRIN domain. Recent data have suggested that ASC functions as an adapter protein linking various PAAD-family proteins to pathways involved in NF-kB and pro-Caspase-1 activation. We present evidence here that the role of ASC in modulating NF-kB activation pathways is much broader than previously suspected, as it can either inhibit or enhance NF-kB, depending on cellular context. While co-expression of ASC with certain PAAD-family proteins such as Pyrin and Cryopyrin synergistically increases NF-kB activity, ASC has an inhibitory influence on NF-kB activation by various pro-inflammatory stimuli, including **TNFa**, IL-1b, and LPS, as measured by NF-kB reporter-gene assays, analysis of expression of NF-kB-responsive genes ICAM and TRAF 1, and by electro-mobility shift assays (EMSA). Gene transfer-mediated increases in full-length ASC or of a mutant containing only the PAAD/PRYIN domain suppressed activation of IkB kinases (IKKs) in cells exposed to pro-inflammatory stimuli, as measured by in vitro kinase assays and immunoblotting using phospho-specific antibodies. Conversely, reducing endogenous levels of ASC using **siRNA** enhanced **TNFa**- and LPS-induced degradation of the IKK substrate, IkBa. Using co-immunoprecipitation assays, we also observed association of endogenous ASC with the IKK complex, suggesting direct regulation of this kinase complex involved in triggering IkB degradation, thereby releasing NF-kB. Our findings thus suggest that ASC modulates diverse NF-kB-induction pathways by acting upon the IKK complex, implying a broad role for this and similar proteins containing PAAD/PYRIN domains in regulation of inflammatory responses.

L7 ANSWER 5 OF 5 CA COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 138:380932 CA
TITLE: TAK1 is Critical for IκB Kinase-mediated Activation of the NF-κB Pathway
AUTHOR(S): Takaesu, Giichi; Surabhi, Rama M.; Park, Kyu-Jin; Ninomiya-Tsuji, Jun; Matsumoto, Kunihiro; Gaynor, Richard B.
CORPORATE SOURCE: Harold Simmons Cancer Center, Department of Medicine, Division of Hematology-Oncology, University of Texas Southwestern Medical Center, Dallas, TX, 75390-8594, USA
SOURCE: Journal of Molecular Biology (**2003**), 326(1), 105-115
CODEN: JMOBAK; ISSN: 0022-2836
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cytokine treatment stimulates the IκB kinases, IKKα and IKKβ, which phosphorylate the IκB proteins, leading to their degradation and activation of NF-κB regulated genes. A clear definition of the specific roles of IKKα and IKKβ in activating the NF-κB pathway and the upstream kinases that regulate IKK activity remain to be elucidated. Here, we utilized small interfering RNAs (**siRNAs**) directed against IKKα, IKKβ and the upstream regulatory kinase TAK1 in order to better define their roles in cytokine-induced activation of the NF-κB pathway. In contrast to previous results with mouse embryo fibroblasts lacking either IKKα or IKKβ, which indicated that only IKKβ is involved in cytokine-induced NF-κB activation, we found that both IKKα and IKKβ were important in activating the NF-κB pathway. Furthermore, we found that the MAP3K TAK1, which has been implicated in IL-1-induced activation of the NF-κB pathway, was also critical for **TNF.alpha.**-induced activation of the NF-κB pathway. **TNF.alpha.** activation of the NF-κB pathway is associated with the inducible binding of TAK1 to TRAF2 and both

IKKα and IKKβ. This anal. further defines the distinct in vivo roles of IKKα, IKKβ and TAK1 in cytokine-induced activation of the NF-κB pathway.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT